

# **Pneumomediastinum is induced during glossopharyngeal insufflation in breath-hold divers.**

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**Abstract:**

*In breath-hold diving as in ordinary scuba diving, there are reported incidents of serious neurological symptoms, mimicking cerebral insults. The etiology for the symptoms in breath-hold diving is uncertain. It has been speculated that it may be due to decompression sickness, provided that there is enough nitrogen in tissue to cause supersaturation. However, as far as we are aware of, such venous gas bubbles have not been demonstrated. Neurological symptoms during breath-hold diving may also be consistent with arterial gas embolism (AGE) caused by gas entering the arterial circulation due to pulmonary barotrauma. The divers often use a special respiratory maneuver to hyperinflate the lungs before diving, termed glossopharyngeal insufflation (GI). After a maximal inspiration the diver opens the glottis and uses the glossopharyngeal musculature to force air into the lungs repeatedly. We hypothesized that this maneuver may increase pulmonary pressure to a degree which overdistends the lungs and causes rupture of the alveoli, thereby being a substrate for AGE. Six competitive breath-hold divers were examined for pneumomediastinum and AGE, by performing computer tomography thorax and transthoracic echocardiography during ongoing GI. Massive amounts of pneumomediastinum were present in four out of six divers. The CVP increased from 0 to a maximum of 28 mmHg, with a mean of 21 at 120seconds apneatime. We conclude that the GI maneuver itself may elevate pulmonary pressures to an extent that may cause pulmonary barotraumas and alveolar rupture. This is a plausible substrate for AGE and subsequent development of neurological symptoms in breath-hold diving.*

## **Introduction.**

Competitive breath-hold diving has become increasingly popular. The sport is frequently associated with serious neurological complications mimicking cerebral insult (1) with symptoms like dizziness, nausea and hemiparesis. Similar symptoms are known from ordinary scuba diving and may then be caused either by decompression sickness (DCS) due to venous nitrogen bubbles or by arterial gas embolism (AGE) due to pulmonary barotrauma. In breath-hold diving, however, the etiology for the symptoms is more uncertain. It has been speculated that it may be due to DCS (2,3), as nitrogen that remains in the lungs from the breath taken before immersion may cause supersaturation. It has also been speculated that during repetitive dives or very deep dives, high levels of nitrogen may be absorbed by the tissues, which may induce supersaturation (4,5). However, neurological symptoms have also been reported in breath-hold diving after only one dive (5,6). Venous gas bubbles should be detectable by echocardiography. However, as far as we are aware of, such venous gas bubbles have not been demonstrated by this method in breath-hold diving (7).

Neurological symptoms during breath-hold diving may also be consistent with AGE caused by gas entering the arterial circulation due to pulmonary barotrauma. In scuba diving this may occur during ascent, due to expansion of the breathing gas. Breath-hold divers do not inhale under water and are therefore not subjected to this mechanism of AGE. However, they often use a special respiratory maneuver to hyperinflate the lungs before diving, termed glossopharyngeal inhalation (GI). After a maximal inspiration the diver opens the glottis and uses the glossopharyngeal musculature to force air into the lungs repeatedly, a movement resembling swallowing (4,8-10). This hyperinflation procedure is popularly called “lung packing” among divers. The purpose is to start the dive with very large lung volumes to reach as deep as possible before the chest and lungs are compressed to the limit of what is tolerable. Additionally, the volume of gas serves as supplemental oxygen storage.

We hypothesized that the hyperinflation maneuver during GI may increase pulmonary pressure to a degree which overdistends the lungs and causes rupture of the alveoli, thereby being a substrate for AGE. To test this hypothesis we examined six breath-hold divers for pneumomediastinum and AGE, by performing computer tomography (CT) thorax and transthoracic echocardiography (TTE) during ongoing GI.

## Methods

### *Test subjects*

The study was approved by the regional committee for medical and health research. Written informed consent was obtained. Ten experienced breath-hold divers were planned to be included. However, the study was terminated after inclusion of six subjects due to high incidence of pneumomediastinum. Inclusion criteria were healthy breath-hold divers >18 years and able to perform GI. Divers having performed GI the last 7 days were excluded. Characteristics of the test subjects are shown in table 1.

**Table 1.** *Characteristics of the test subjects.*

	# 1	# 2	# 3	# 4	# 5	# 6
Height (cm)	195	173	176	183	183	179
Weight (kg)	98	78	80	83	85	72
Age (years)	32	26	29	28	41	26
History of smoking	No	No	No	No	No	No
Constant weight (meters)	60	20	67	84	60	25
Free immersion (meters)	50	-	-	100	-	-
No limit (meters)	50	-	-	103	-	-
Max apneatime (min:s)	6:09	4:05	6:13	7:04	6:45	4:15
Former neurological symptoms after breath-hold diving	No	No	No	*1	*2	No

\*1: Numbness in the right arm and right leg after an 80 meter dive.

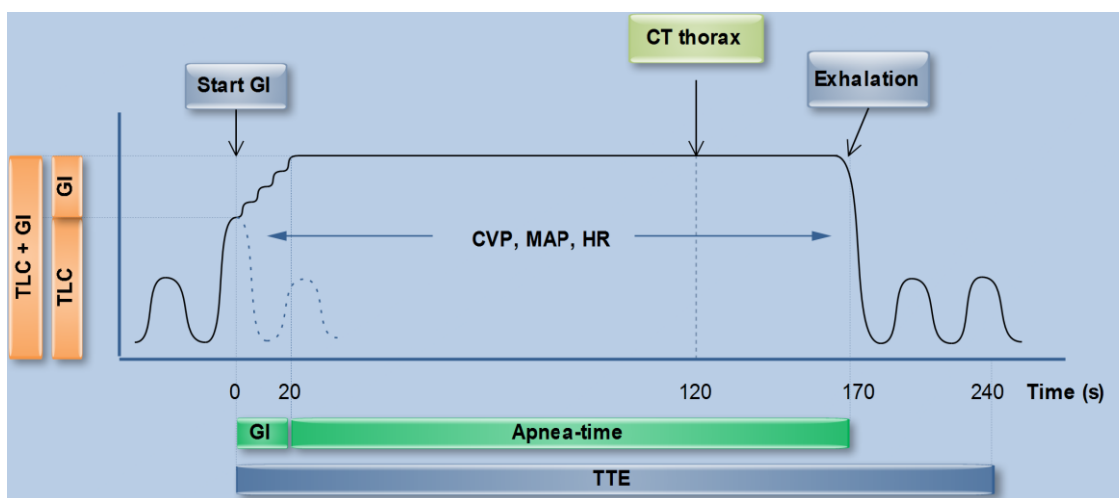
\*2: Loss of motor control after a 60 meter dive.

### *Monitoring*

A 20G arterial catheter was inserted in a radial artery to measure systemic invasive blood pressure. A 16G line (Cavafix, Braun ®) to measure central venous pressure (CVP) was inserted via the basilican vein in the elbow and located at the entrance of the right atrial chamber. Correct positioning was verified by a CT prescan. Arterial oxygen saturation (SpO<sub>2</sub>), and cardiovascular parameters were monitored throughout the experiment. A 5-lead electrocardiogram (ECG) was continuously monitored throughout the experiment.

### *Experimental procedure*

Experiments were conducted from May 2009 to October 2009, with three anesthesiologists experienced in hyperbaric medicine present. The hospital multiplace hyperbaric chamber was located close to the laboratory and ready for use if a gas embolus was suspected. Spirometry, hemodynamic measurements, CT thorax and TTE were performed as illustrated in figure 1, with the test persons in the supine position. Variables measured were vital capacity (VC), the volume packed by GI, CVP, heart rate (HR) and mean arterial pressure (MAP). Data on CVP, MAP and HR were obtained every 10 s.



**Figure 1.** Illustration of the experimental procedure.

The steps represent separate boluses of air insufflated during glossopharyngeal insufflation (GI) on top of the total lung capacity (TLC). GI was finished within 20 seconds. The test subjects then held their breath (apnea-time) and exhaled at 170 seconds. Transthoracic echocardiography (TTE) started at time of inspiration and continued for one minute after exhalation. Central venous pressure (CVP), mean arterial pressure (MAP) and heart rate (HR) were monitored continuously from start of inspiration to exhalation. CT thorax started after 120 s apnea.

### *Spirometry*

In order to achieve large lung volumes and high intrathoracic pressures, the test subjects were asked to inhale to maximal lung volume and then employ GI. Vital capacity (VC) and the amount of air “packed” by GI were measured. Lung volumes were measured during exhalations from VC and from VC with the addition of GI (VC+GI). The difference was used to calculate the amount of gas insufflated during GI. In order to perform and measure GI by spirometry the subjects had to remove the mouthpiece when performing the GI (nose clip in place). Subsequently, without air leak, they returned the mouthpiece and exhaled to residual volume (11).

### *CT thorax*

Scanning was performed using a 64-slice scanner (Brilliance 64, Philips Medical Systems). Following a prescan, test subjects performed GI and held the breath for 170 s. A non-contrast-enhanced scan was then performed in all test subjects using a low radiation dose protocol. The scan started after 120 s, while the test subjects still held their breath, beginning at the thorax aperture and ending at the level of the diaphragm (typically 27 cm). Scan parameters were: Tube rotation speed 750 ms, detector collimation 64 x 0.625, table pitch 0.8, tube voltage 120 kV, tube current 30 mAs / slice, reconstruction image matrix 512 x 512, section thickness was 0.67 mm with an increment of 0.33 mm. CTDI volume was 1.8 mGy / cm, DLP was 62 mGy (27 cm). Estimated effective whole body dose was 1.2 – 1.5 mSv.

### *TTE*

TTE (Vivid-i, GE Medical Systems. Probe 3S-RS, GE Medical Systems) was recorded continuously during the entire breath-hold period by an experienced investigator. The echo-probe was placed substernal and recordings started before GI and ended one minute after the breath-hold period. The CT-scanner was in position and set to scan at any time a bubble was detected. The investigation focused on the left atrium and left ventricle. However, all four chambers were viewed most of the time. Recordings were taped for later analysis.

## Results

All test persons described difficulties in holding the breath during apnea due to the high intrathoracic pressure. After 60 s test subject # 4 had to release air, which caused a drop in CVP. SpO<sub>2</sub> was >98 % in all test subjects during apnea.

### *Spirometry*

All test subjects had VC within or above the normal range and GI increased lung volumes by 1.4 l (range 0.8 – 2.2 l). Lung volumes for the individual test subjects are given in table 2.

**Table 2.** *Vital capacity, the volumes added by glossopharyngeal inhalation (GI), occurrence of pneumomediastinum and symptoms for the individual test subjects.*

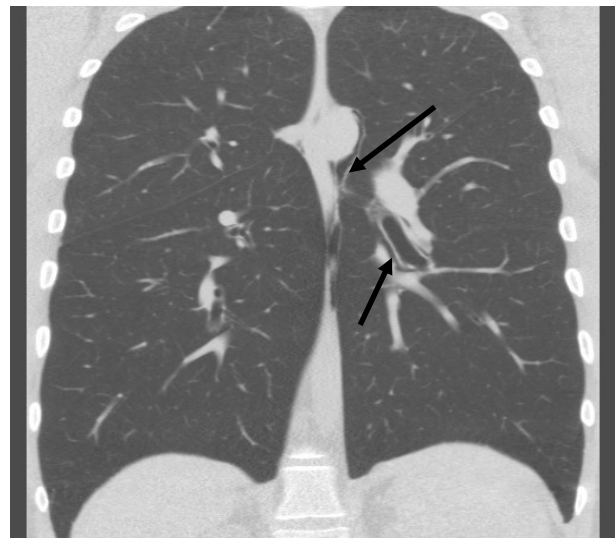
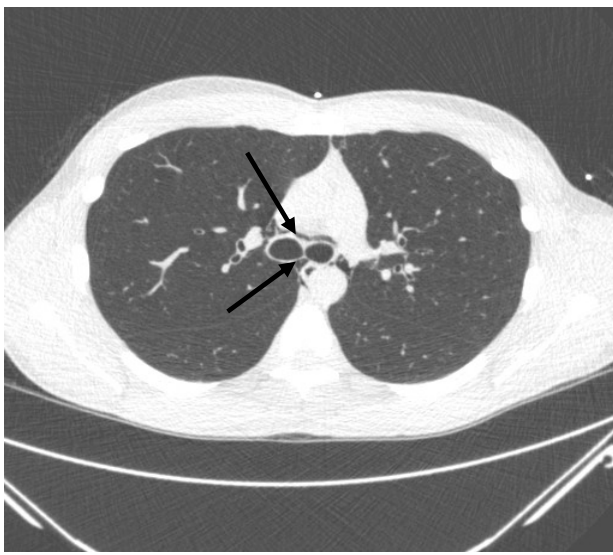
	# 1	# 2	# 3	# 4	# 5	# 6
Vital capacity (l)	6.0	6.3	5.3	6.7	5.9	5.3
Vital capacity + GI (l)	7.4	7.1	7.0	8.9	7.3	6.3
GI (l)	1.23	0.8	1.7	2.2	1.4	1.0
Difference (%)	21%	12%	32%	33%	23%	19%
Pneumomediastinum	Yes	No	Yes	Yes	No	Yes
Symptoms during the study	No	No	No	*1	No	*2

\*1: Loss of consciousness 60 s after GI on first attempt.

\*2: Intense chest pain and respiratory pain lasting two days.

### *CT thorax*

CT thorax at 120 s after start of GI revealed pneumomediastinum in four of six test subjects. This is demonstrated by findings from # 1 and # 4 in figure 2. Subject # 4 also had a minor medial pneumothorax.



**Fig 2.** Computer scanning of test subject # 1(left) and # 4 (right). Arrows indicate air in the mediastinum.

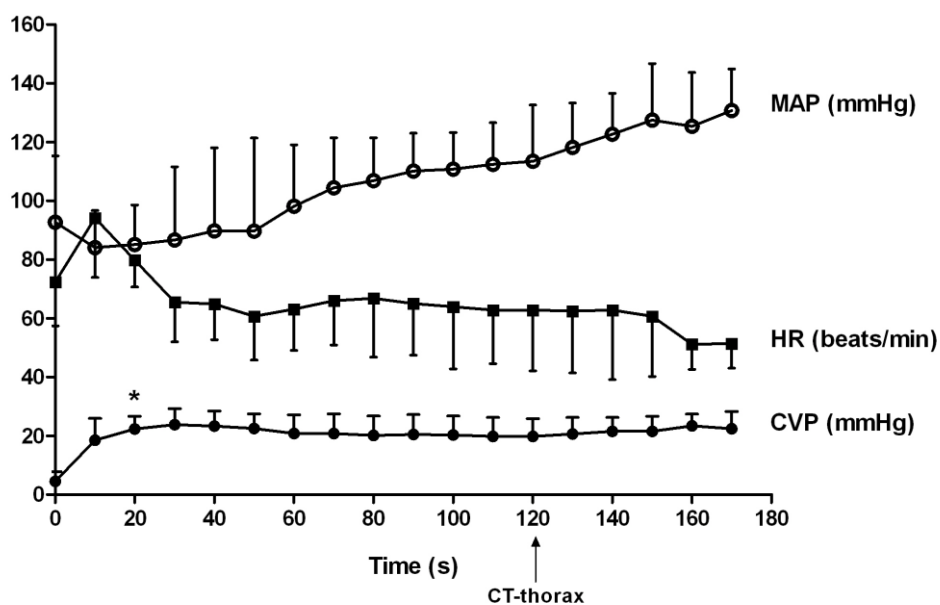
### *TTE*

TTE reveal no intravascular air bubbles at any time point in any of the test subjects.

### *Hemodynamics*

CVP, MAP and HR throughout the experimental procedure are shown in fig 3. During the GI maneuver CVP increased rapidly from  $4 \pm 3$  to  $22 \pm 4$  mmHg and then remained above 20 mmHg during the apnea period. A trend towards a reduction in MAP was seen during the period the diver performed GI ( $93 \pm 23$  vs  $85 \pm 13$  mmHg). MAP then increased continuously reaching  $131 \pm 14$  mmHg at time of exhalation. A trend towards a rapid increase in HR was seen during the GI manoeuvre ( $72 \pm 15$  to  $94 \pm 20$  beats/min). HR then gradually declined to  $51 \pm 8$  beats/min.

**Fig 3.** Mean arterial pressure (MAP), heart rate (HR) and central venous pressure (CVP) from start of inhalation ( $t = 0$ ) until exhalation ( $t = 170$ ).





### *Symptoms*

One test subjects with pneumomediastinum (# 6) experienced intense chest pain and respiratory pain for two days after the experiment. He did not develop any neurological symptoms and was not in need of medical treatment. Chest x-ray taken the next day was normal. Test person # 4 experienced a brief loss of consciousness 60 s after having performed GI. He quickly regained consciousness after exhaling.

## Discussion

The main finding in the present study is that pneumomediastinum, an objective indication of alveolar rupture, was found on CT thorax in four of six test subjects (67%) during ongoing GI. This finding is in accordance with our hypothesis and shows that GI can increase pulmonary pressures to an extent which causes rupture of the alveoli, a potential substrate for AGE.

It is a well-known fact that high intrapulmonary pressure may cause pulmonary barotrauma, and pneumomediastinum has been observed in mechanically ventilated patients. (12). Therefore, the airway pressure is kept as low as possible. However, AGE due to barotrauma has, to our knowledge, not been observed. In the present study we have shown that the GI maneuver elevated pulmonary pressure to an extent, which caused pulmonary barotraumas, visualized by pneumomediastinum on CT thorax. However, we did not observe AGE. The physiological explanation may be that during GI, blood is removed from the thorax causing the vessels to collapse (13). Hence, air is prohibited from entering the vessels. However, during breath-hold diving, the gas in the thorax is compressed and due to the underpressure the vessels fill with blood. This may cause a situation where alveolar gas migrates through the ruptures and enter into the bloodstream. In our study the most experienced divers reached the highest VC during GI. They are, however, not necessarily the ones who succumb to pneumomediastinum. This is probably a result of the elasticity in the lung tissue. Regular overexpansion (read: training) may increase this elasticity (14).

Pulmonary barotrauma induced by GI has previously been suggested in breath-hold divers (4,9,15-17), but there are only two reports in which pneumomediastinum actually have been demonstrated (14,16). Pneumomediastinum does usually not cause great symptoms and is therefore probably overlooked. In 2006 Jacobson et al (16) presented one case report of pneumomediastinum after lung packing. The diver participated in a study with both glossopharyngeal insufflation and glossopharyngeal exhalation (14). The pneumomediastinum was seen on CT-scan and the authors concluded that pulmonary overdistension caused it. In the present study we used CVP, measured at the entrance of the right atrium, as an estimate of pulmonary pressure. The four test subjects who experienced pneumomediastinum had maximal CVP from 25 to 31 mmHg. This is comparable to the case report by Jacobsen et al (16) where transpulmonary pressure was 22 and 31 mmHg during GI measured on two occasions, using an esophageal balloon-catheter. The present study is, as far as we are aware of the first to actually demonstrate pneumomediastinum during ongoing GI. None of the test subjects had any preexisting pulmonary symptoms or had performed GI the last 7 days, so that preexisting pneumomediastinum was highly unlikely.

There are many reports on breath-hold divers who have experienced neurological symptoms (1-3,5,6,15,18-21). It may be difficult to distinguish between DCS and AGE because symptoms may be similar. The most important factor is time of onset for symptoms. Symptoms experienced within 10-15 minutes of surfacing are most likely caused by AGE (22, 23). The onset of DCS is slower and becomes symptomatic in 90% of patients within 6 hours (24, 25). Neurological symptoms related to breath-hold diving vary widely in explicit

information of events and findings. In some reports hyperventilation prior to diving is reported, but there is no information on use of GI (5,15).

Kohshi et al have presented several case reports in which neurological manifestations are accompanied by MRI abnormalities in Japanese Ama divers. In an interview survey among Ama divers, 13 out of 16 had a history of neurological complications related to breath-hold diving and 9 of these had symptoms mimicking cerebral insults during diving or within 10 minutes after surfacing (15,21). Time to onset of symptoms in the remaining 4 is not documented. The authors concluded that venous bubbles shunting through to the arterial circulation caused the symptoms. However, the symptoms might as well be explained by AGE due to alveolar rupture. Fitz-Clarke published a survey of the risk of DCS in breath-hold dives beyond 100 meters (5). It was not documented if GI was used, but these depths are only possible to reach by performing GI prior to the dive. In the material of 192 dives (24 divers), two divers died during ascent and two other underwent treatment for what was believed to be DCS. One of them had severe vertigo when appearing at the surface. He also rapidly developed hemiparesis. Due to the rapid onset of symptoms, these serious events are also consistent AGE. Wong (26) reported two cases where breath-hold divers developed visual blurring and dizziness during diving. The author does not discuss if the symptoms are believed to be due to DCS or AGE, but both individuals had symptoms and time to onset consistent with AGE.

If one assumes that symptoms are caused by DCS, one should be able to detect venous bubbles in breath-hold divers by echocardiography (27). However, as far as we are aware of, such venous gas bubbles have not been demonstrated by this method in breath-hold diving. Boussuges et al (7) studied top-level breath-hold divers during five hours of repetitive diving at depths from 28 to 40 m and did not detect circulating venous gas bubbles by continuous cardiac ultrasound. These data support our interpretation that AGE due to alveolar rupture is a more likely cause of the neurological symptom in breath-hold divers than DCS.

## **Conclusion**

We conclude that the GI maneuver itself may elevate pulmonary pressures to an extent which in four of six individuals caused pulmonary barotraumas and alveolar rupture, visualized by pneumomediastinum and pneumothorax at CT thorax. However, we did not observe AGE. Alveolar rupture and pneumomediastinum during the GI maneuver is a plausible substrate for AGE and subsequent development of neurological symptoms in breath-hold diving.

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